Asbestos Timetables – A guide for policymakers

(Basic History of Usage, Knowledge & Disease Associations: Chronologically up to the approximate time of the passage of the Occupational Safety and Health Act of 1970 in the United States accompanied by up-to-date notes on selected diseases, fiber types, industries, non-occupational, occupational, and occupational guidelines and standards for exposures to asbestos)[1]

By
Richard A. Lemen, Ph.D.[2]

"In the great civilizations of antiquity, whether in the East, West, or in Europe generally, there was sufficient concentration of the forces of labour to produce the intensest forms of the maladies classed by Pliny as the "diseases of slaves." Some of the most injurious processes known to us now are extremely ancient. To mention but a few:. the textile processes of preparing and weaving asbestos and flax."

Adelaide M. Anderson, H.M. Principal Lady Inspector of Factories. Chapter II. Historical Sketch of the development of Legislation for Injurious and Dangerous Industries in England. In: Dangerous Trades, Ed. Thomas Oliver, John Murray, London, 1902.

Chapter 1:

Usage and Production

I
History of Asbestos Usage

"Asbestos is one of the most marvelous productions of inorganic nature. It is a physical paradox, a mineralogical vegetable,* both fibrous and crystalline, elastic and brittle; a floating stone, as capable of being carded, spun, and woven, as wool, flax, or silk."
"Occupying the apparent position of a connecting link between the mineral and vegetable kingdom, it would appear to possess some of the characteristics of both, while being altogether different from either."

R. H. Jones, 1897. Asbestos and Asbestic – Their properties, occurrence, and use. Crosby Lockwood and Son, London, p. 1

---

[1] This document is not intended to be all inclusive nor to discuss every event or article relative to asbestos. It is, however, intended to give the reader the highlights and a basic history of the usage, knowledge & disease associations demonstrated or suspected to relate from exposures to asbestos, either in the workplace or away from the workplace. There are multiple books demonistrating or investigating the asbestos industry and the causation of diseases from exposures associated with asbestos. This author's intent is to educate the reader, by this evaluation and interpretation of the history of asbestos, which is the sole purpose of this treatise.

2500 B.C	The use of asbestos dates back thousands of years when asbestos fibers were being incorporated into pottery (Agricola, 1556; Noro, 1968).
1824 A.D.	Discovery of asbestos in the United States near Lowell Vermont, but not much interest was shown until the 1890s (____, 1921).
1879	Canadian chrysotile mine opens in the Province of Quebec (Jones, R.H., 1897).
1880	*****[3]The modern industry dates from about 1880, when asbestos was used to make heat and acid resistant fabrics (Hendry, 1965; and Hueper, 1942; 1965).
1886	In Osaka, the first Japanese asbestos factory opens making packing and other insulation products (Morinaga et al., 2001).
Early 1888s	Crocidolite, meaning "woolly stone", asbestos was first found northwest of the Cape Province of South Africa, but was not actively mined until the demands for asbestos during World War II (Cilliers and Genis, 1961; Hall, 1930; Sleggs et al., 1961; and Sluis-Cremer, 1970).
1895	In England a patent was awarded for railroad brake linings containing asbestos (Raybestos-Manhattan, 1968).
1903	Friction brake products sold in the United States (Raybestos-Manhattan, 1968).
@ 1904	Second deposit of asbestos was found in South Africa in northeast Transvall and was named amosite, in 1918, from the village Amosa, which was the acronym for the term Asbestos Mines of South Africa. Production began in the mid-1920s, by Cape Asbestos Company the same company already mining and producing crocidolite (____, 1948; and Sluis-Cremer, 1970).
1/13/06	Johns-Manville ran full page advertisements in The Saturday Evening Post saying [asbestos] *"Serves More People in More Ways than any Institution of its kind in the World."* Included in this ad were products for the home builder, the industrial and commercial builder and the automobilists. Asbestos was also being used by American steel companies for insulation of large furnaces (Cirkel, 1910).

II
Highlights in the production history of asbestos

"On its [asbestos] introduction it was looked upon with some degree of suspicion, and only 300 tons were mined during the fist year, which realized no more than $19,500. But in proportion as it became better known the rapidity of its progress was prodigious. By 1890, the output had grown to 9,860 tons, and its saleable value hand reached $1,260,240. This was a grand time for mine owners, when even by straining every nerve, under the stimulus of daily advancing prices, they were unable to supply the demands of the manufactures; while these last daily found increasing difficulty in their endeavours to obtain adequate supplies of the raw material to meet their requirements. No forward contracts could be made, and it was impossible to foretell to how high a figure prices would eventually reach."

R. H. Jones, 1897. Asbestos and Asbestic – Their properties, occurrence, and use. Crosby Lockwood and Son, London, Preface[4]

---

[3] ***** **represents major historical events.**

1866	Asbestos used as heat insulation (Bowles, 1937)
1870	Asbestos cement used as a boiler covering in 1870 (____, 1903)
1871	Opening of the first asbestos factory in Great Britain (____, 1953)
1874	Commercial production of asbestos insulation materials in 1874 (____, 1903)
1890	The first processing of Canadian asbestos into textile in the U.S. (Berger, 1963)
@ 1890	The asbestos cement pipe industry had its origins in Italy (____, 1973)
1903	Asbestos cement production in the U.S. began in 1903 (Berger, 1963)
1904	Flat asbestos cement board was produced in the U.S. in 1904 (____, 1958)
1906	Asbestos was first used as a brake lining in 1906 (____, 1953)

[4] **Notes on Asbestos Uses:** Often asbestos is referred to as the "magic Mineral" having 3000 or more uses, including such uses as being woven into cloth, with vegetable fibers, to still the sound of falling trees during construction projects, within the Roman empire; for wrapping the corpuses, referred to by Pliny as the funeral dress of kings, prior to cremation in order to help collect the ashes; in making clay pots some 4000 years ago; and was even mentioned by Marco Polo, during his travels to the far east, where he find it called "salamander" which was mined from the mountains, extracted then crushed, by subjects of the Great Khan, into a fibrous like wool that was then spun and made into cloth of which some were used for table cloths, that when soiled, were thrown into the fire and came out "white as snow" for use again; one was sent to the Pope, in Rome, "in which cloth he keeps the Sudarium of our Lord." (Jones, 1897, p. 5). Also, Pope Clement the Eleventh ordered an intact shroud of considerable length, in good condition and as pliant as silk, found in a sarcophagus by the Via Praenestina in 1702, a road to the very ancient city of Latium, lying 23 miles east of Rome, placed in the Vatican library where it can still be seen (Jones, 1897). Strabo and Plutarch have both mentioned the use of asbestos for wicks used in the lamps of the Vestal Virgins as well as being used for sacred fires in the Temples, being referred to as perpetual since the flames do not consume the wicks or the asbestos placed in the fires (Jones, 1987, p. 7). Charlemagne used the "amianthine" (asbestos) table cloths to astonish his rude warrior guests, throwing them into the fire then withdrawing it cleansed and unconsumed (Jones, 1897, p. 10). At the Royal College of Surgeons in England, the oldest mummy in the world, upwards of 6,000 years old, was unwrapped by a Professor Stewart who found the body wrapped in gauze-like material and the cavities of the body stuffed with the same type material, which later was identified as a linen like material thought to be made of asbestos fibers (Jones, 1987, p. 9). Tribes of Indians were known to have made dresses of asbestos, "which they cleanse by throwing them into fire." (Jones, 1897, p. 18). Benjamin Franklin even bought a purse from the "northern part of America" made from woven tremolite asbestos, a picture of which is found in the book by Selikoff & Lee (Latham, 1958; Liddell & Miller, 1991& Selikoff & Lee, 1978). Giuseppe della Corona, a Florentine priest is credited with the introduction of asbestos millboard in the mid-1800s (Jones, 1897, p. 15). A unique use can be found in John Baxter's book **A Pound of Paper**, in which he discussed the use of Johns-Manville Quintera, a form of asbestos, to cover a limited edition of author Ray Bradbury's book Fahrenheit 451, published in 1953 (Baxter, 2003). The manufacture of asbestos-paper dates back to around 1700, when it was made in Norway and for printing banknotes and other securities in Italy, in the mid-1800s (Jones, 1897, p. 14 & 15). More in-depth descriptions of the ancient uses of asbestos can be found in the first chapter of the book written by Robert H. Jones, a mineralogist, published in 1897 and also in discussions of the Magic Mineral by Paul Brodeur (Jones, 1897 & Brodeur, P., 1968 & 1972).

1928 The first pipe making machines were imported into the U.S. (Berger, 1963)

1944 Asbestos spraying of deckheads and bulkheads begun in British Navy Ships (Harries, 1968; and Harries, 1971).

1963 The spraying of British Navy ships with asbestos was discontinued (Harries, 1968; and Harries, 1971).

Chapter 2:

Knowledge of The Health Effects From Exposure To Asbestos:

"In the great civilizations of antiquity, whether in the East, West, or in Europe generally, there was sufficient concentration of the forces of labour to produce the intensest forms of the maladies classed by Pliny as the "diseases of slaves." Some of the most injurious processes known to us now are extremely ancient. To mention but a few: . . . weaving asbestos and flax."

Adelaide M. Anderson, 1902: Historical sketch of the development of legislation for injurious and dangerous industries in England. In: Dangerous Trades. Ed. Thomas Oliver, John Murray, London

I
Lung Disease[5]

61-114 A.D. Pliny the Younger wrote of the illness of slaves who worked with asbestos (Anderson, 1902 & Selikoff & Lee, 1978).

1899 Lucy Dean, Women Inspector of Factories, writes in the 1899 Annual Report on the Health of Workers for 1898, that "[T]he evil effects of asbestos dust have instigated a microscopic examination of the mineral dust [asbestos] by HM Medical Inspector. . ., the effects have been found to be injurious as might

[5] **Notes on Asbestosis:** Asbestosis is a chronic lung disease due to the inhalation of asbestos fibers, either of the amphibole or serpentine type, and is characterized by diffuse interstitial fibrosis and frequently is associated with pleural fibrosis or pleural calcification. X-ray changes are usually small irregular opacities occurring mainly in the lower and middle lung fields. The pulmonary fibrotic changes develop slowly over the years---often progressively, even without further exposures---and their radiographic detection is a direct correlate of their extent and profusion. In some cases, minor fibrosis with considerable respiratory impairment and disability can be present. Pulmonary hypertension is frequently associated with advanced asbestosis and the resultant cor-pulmonale (right-sided heart failure) may be the cause of death. In some cohorts this has accounted for 12 to 20% of the deaths (Kleinfeld et al., 1967 and Krige, 1966). Asbestosis is a progressive disease even in the absence of further exposure (OSHA, 1986). Individuals diagnosed with pulmonary asbestosis are at a higher probability of developing and dying of cancer of one or other forms (HMSO, 1949; Buchanan, 1965; O'Donnell, et al., 1966; Lewinsohn, 1974; and Berry, 1981). Nine member clinics, from the Association of Occupational and Environmental Clinics (AOEC), reported seeing 2057 patients between 1997 and 2000 for asbestos-related conditions, 95% of whom were diagnosed with asbestosis/parenchymal disease principally in the construction (SOC code 63-64); production working occupations i.e. welders, labors, machine operators etc. (SOC 71, 73-78) and the handlers, cleaners, helpers & laborers (SOC 86-87) occupational categories (Hunting & Gavitt, 2003). Most researchers believe that asbestosis is linearly related to cumulative exposure and because very low concentrations of asbestos do not result in radiological, pathological or clinical evidence of lung fibrosis suggests there may well be a threshold for asbestosis (Karjalainen, 2002).

have been expected." She continues "the worker can continue for a very long time apparently unaffected, before the symptoms of the evil become marked." (Dean, 1899).

1902 Adilaide Anderson, Lady inspector of Factories included asbestos among the dusts known to cause injury to man in a publication on dangerous industries in England (Anderson, 1902).

1906 *****The first recorded case of asbestosis was reported, in London, in a 33 year old man who worked in an asbestos textile plant for 14 years, by a Charing Cross Hospital physician Dr. Montague Murray in 1906 (Murray, 1907).

1906 Numerous deaths (@50) reported in a French asbestos textile factory (Auribault, 1906).

1908 Italian physician reviewed the cases of 30 asbestos workers seen in a Turin clinic between 1894-1906, having a serious pulmonary disease, thought to be tuberculosis, however, which was extremely progressive, unlike the typical tuberculosis case (Scarpa, 1908).

1911 Reported in the Annual Report of HM Chief Inspector of Factories for 1910 that a Professor J.M. Beattie, of Sheffield University in the UK, had shown mild degree of fibrosis in experimental animals after inhalation of asbestos containing dust & that 5 deaths of persons with phthisis among a workforce of less than forty in the production of woven asbestos. Dust suppression and prevention though ventilation was recommended to protect workers from disease (Collis, 1911).

1912 The American Association for Labor Legislation mentioned asbestos related disease in their Industrial Diseases (____, 1912)

1912 The government of Canada Department of Labour included asbestos related diseases as an industrial disease (____, 1912).

1914 Case report of a woman having worked in a German asbestos factory and dying of an acute lung illness resembling pleural pneumonia, however, on autopsy there were ". . . large number of crystals of a peculiar nature" (Fahr, 1914). This was the 1st case reported in Germany and was presented to the medical society of Hamburg.

1918 *****The first description in the medical literature on x-ray changes in 15 individuals exposed to asbestos (Pancoast et al., 1918).

1918 It was reported in the *Bulletin of US Labor Statistics* that American and Canadian, insurance companies would not insure asbestos workers due to the un-healthy conditions in the industry (Hoffman, 1918)

1924 Case report of asbestosis, with extensive fibrosis and more so in the right lung, in a 33 year old woman, **Nellie Kershaw**, who had worked in asbestos factories for 20 years. This case had pleural thickening over the entire surface of the lung with dense adhesions on the chest wall and the pericardium. There was also tuberculosis lesions present (Cooke, 1924).

1925 Pancoast & Pendergrass published their review of the present knowledge on the

pneumoconiosis[6], including asbestosis, in *The American Journal of Roentgenology and Radium Therapy*, a journal read by mainstream general medicine radiologists (Pancoast & Pendergrass, 1925).

1927 *****First complete description of asbestosis, including the naming of the disease and a description of *"curious bodies"*, observed in lung tissue (Cooke, 1927).

1927 Description of asbestosis observed in lung tissue, appearing first in 1924 (Cooke, 1924) and three years later in 1927 (McDonald, 1927; Cooke, 1927; Cooke and Hill, 1927; and Oliver, 1927). Also, McDonald (1927) and Cook & Hill (1927) described peculiar bodies in the lungs now known as asbestos bodies.[7]

1927 First official claim for asbestosis reported in the United States was filled in Massachusetts in a foreman in the weaving department of an asbestos plant and a fatal case of uncomplicated asbestosis was reported to the Medical Society of South Carolina (Lanza, 1936).

1928 In May, 4 cases of asbestosis were reported, one case having only 2 years of exposure to asbestos and having no histological evidence of tuberculosis. The study report stated that it had been known for sometime that workers exposed to asbestos materials suffer from pulmonary disabilities (Simson, 1928). One case, a South African asbestos mill worker, was only exposed 12 months died of rapid TB and on autopsy was found to have moderate fibrosis. Simson also reported that if due to asbestos dust was much more rapid that the fibrosis produced by silica.

1928 *****The Journal of the American Medical Association ran an editorial on pulmonary asbestosis in January because they felt that the dangers of asbestosis and its unique pathologic features deserved more attention than had been given to the disease (JAMA, 1928).

1928 On December 1, a case report of fibrosis in a 40 year old man, who had worked in the asbestos industry for 22 years, was published in which all other causes were excluded including tuberculosis (Seiler 1928).

1929 Cooke and Gloyne independently describe curious bodies found in pulmonary asbestosis (Cooke, 1929 & Gloyne, 1929).

1929 Stewart & Haddow (1929) demonstrate asbestos bodies in the sputum of asbestos workers.

1929 An article in the British Medical Journal reviewed occupational induced dust diseases, including asbestos related disease and stated that the "curious bodies" were an indication of exposure to asbestos (Bridge, 1929).

1930 The first reported case of asbestosis in the United States was reported in Minnesota of a man who had worked previously in a South American asbestos mine starting in 1911 (Mills, 1930).

---

[6] A term meaning dust affecting the lung, taken from Zenker's orginal term pneumonokoniosis (Meiklejohn, 1956).

[7] Today it has been shown that asbestos bodies can form in extrapulmonary sites, such as the liver and spleen (Williams et al., 2001).

1930 Lynch and Smith (1930) reported on "asbestos bodies" found in the sputum of asbestos exposed workers.

1930 *****Five additional early studies led many investigators to conclude that people exposed to asbestos dust, including manufacturing, developed the disease "asbestosis" (Merewether and Price, 1930; Merewether, 1930; Wood and Gloyne, 1930; Wood and Page, 1930; Soper, 1930). Merewether and Price (1930) in their report found 28.1 percent of the 374 asbestos textile workers examined with pulmonary fibrosis and for those with greater than 20 years exposure, 80% had x-ray abnormalities. Even after excluding any of those with other known or suspected dust exposure history, there remained 26.2 percent with pulmonary fibrosis that could only be explained as a result of their asbestos exposure. Wood & Page (1930) evaluated the case of a 21 year old female with a rapid evolution of tuberculosis exposed to asbestos with asbestosis bodies developing within two years from first exposure and asbestos fibers found in the lungs on post mortem. Soper's case report is of a 30 year old man who began work in an asbestos plant at age 17. Soper reports the most common symptom in pulmonary asbestosis is dyspnoea and that the lung fibrosis is a progressive disease with fibrosis of both lungs and basal pleurisy.[6]

1930 Suppression of dust was recommended to control lung fibrosis, caused by asbestos, in a report issued by HM Chief Inspector of Factories (Merewether & Price, 1930).

1930 Merewether (1930) described the pulmonary fibrosis as affecting the basal region of both lungs and discusses the differences between silicosis and asbestosis. He also discusses the dose-response from exposure and the risk of disease.

1930 *****The Journal of the American Medical association published statistical highlights of asbestosis as reported by Merewether and others and of the other knowledge of asbestosis as well as the introduction of a bill by Lord Russell into the parliament to amend the workmen's compensation act to processes involving exposure to asbestos (JAMA, 1930). The JAMA was mailed, by 1920, to 48% of U.S. doctors but estimated to be read by 80% of U.S. physicians (Fishbein, 1947).

1930 *****The Lancet, the joint American and British medical journal, published an editorial on pulmonary asbestosis discussing the Merewether Price report and others highlighting the need for prevention and recommended prohibiting young persons from working in specially dusty work (Lancet, 1930).

1930 *The Asbestos Worker*, a trade union journal for the asbestos worker made reference to asbestosis (____, 1930).

1931 Asbestos induced discrete pleural thickening (pleural plaques) were first reported by Sparks (1931). He also described small irregular calcareous deposits in the lower parts of the lung. Sparks also concluded that because all his patients came for examination voluntarily, once symptoms appeared, that examination of a group of workers from an asbestos factory was unlikely to discover gross changes, thus questioning the value of cross-sectional screening of the active workforce.

1931 Asbestos exposure studies continued to be reported showing the development of asbestosis (Lynch and Smith, 1931 & Sparks, 1931).

1931 Another study discussed the case of asbestosis in a 10 year old wired-haired terrier used as a ratter in an asbestos factory (Schuster 1931).

1931 Asbestosis, its identification and control discussed in a UK Government publication (UK, 1931).

1932 Asbestosis after asbestos exposure indicating how the x-ray is different than that of silica exposed workers. (Russell, 1932).

1932 The disease asbestosis was causally liked with end-product usage of asbestos containing materials, as early as 1932, when a maintenance employee, working with asbestos containing insulation products, developed the disease. A workers' compensation claim was even awarded, in this case, without any medical challenge (Russell, 1932).

1932 Stewart et al. (1932) discusses clumps of asbestos bodies and their role in the pathogenicity of asbestosis.

1933 Asbestos exposure study and the development of "asbestosis" in 7 cement mixers cases and a dog. This study reported asbestosis cases among production workers and described the slow development of the disease with the patient often free of symptoms for several years thus the latency period is brought in as a part of the etiology of asbestosis. Finger clubbing was discussed as were asbestos corns. Case 1 was in a 22 year old female with only 4 years as a mattress maker. Case 2 was in a 35 year old female asbestos factory worker with 5 years exposure. Case 3 was in a 26 year old card room worker for 6 years with progressive spread of her fibrosis in both lungs in less than 9 months. Case 4 was in a 31 year old female asbestos factory worker making mattresses for 3 years. Case 5 was in a 34 year old female asbestos factory worker for 6 years and seen 13 years later with a progressive cough and dyspnoea and with asbestos corns on the hand and elbow. Case 6 was in a 43 year old man who was a superintendent of the card room for 9 years who first started developing dyspnoea after 4 years to the extent he was forced to quit 5 years later. He had marked clubbing of the fingers. Case 7 was in the case of asbestosis in a 10 year old rough-haired terrier dog used as a ratter in an asbestos factory, that had been reported in 1931 by Schuster, Ellman concludes that pulmonary asbestosis is a progressive disease with a bad prognosis and its treatment can only be symptomatic (Ellman, 1933)[8]. .

1933 Ellman discussed a case of asbestosis in a person exposed to asbestos dust for 10 years which did not entail exposure to high dust concentrations from asbestos insulation used for coating on lead pipes (Ellman, 1933).

1933 Professor Beattie's study of experimental animal reported in HM Annual Report of 1910, said *"Although definite proof was not forth-coming at the time, there were reasonable grounds for suspicion that the inhalation of much asbestos dust was to some extent harmful, and form then onwards the British Factory Department pressed for the installation of exhaust ventilation in the more dusty processes."* (Collis, 1911).[9]

[8] These studies and others followed describing the latent period for disease development where the authors found the longer the duration of exposure the greater the risk of developing disease.

[9] **Notes on Asbestosis in Field Animals:** Asbestosis is not specific to humans and has occurred in animals other than under experimental situations. Besides the terrier described above by Ellman (1933) and Schuster (1931), Webster (1963) described asbestosis in donkeys hauling asbestos ore. Environmentally induced asbestosis has also been found in field rats living in and

1933 Merewether (1933) concludes that exposure to asbestos for a period of less than 5 years can cause asbestosis which can result in death. He emphasizes the prevention of asbestosis is to reduce the concentration of dust.

1934 It was demonstrated that disease occurred even after short periods of exposure and that radiographic changes occurred in workers exposed less than 5 years. He further states that the "dusty trades" have been considered "inimical" [hostile] to the health of employees for a long time. He further states that asbestosis once acquired is definite and a serious industrial hazard which is permanent and more or less rapidly progressive (Donnelly, 1934).

1934 Studies linking asbestos exposure with to asbestosis (Wood and Gloyne, 1934; Donnelly, 1934). Wood and Gloyne (1934) concluded that whether or not tuberculosis is associated with asbestosis it is certainly less than that found with silicosis.

1935 Donnelly reads his findings concerning asbestosis at the American Public Health Association convention held in Milwaukee, WS in October 7 – 10. In the paper he discusses that the dyspnoea is out of proportion to the other symptoms even with no improvement though the X-ray films show a negligible increase of the pathological process as the disease progresses over the years. He also states that the effectiveness of personnel protective devices may be doubtful and that engineers familiar with the asbestos hazard can provide the highest type of protection (APHA, 1935).

1935 Reports linking asbestos exposures with asbestosis are discusses as well and findings of a survey conducted by the state of Pennsylvania starting in 1933. The Department of Labor and Industry was concerned with a lack of information in their state and asked the Asbestos industry in the state to help survey the hazard as pertains to dustiness and physical condition of the workers. They found that in counting all particles that those particles less than 10 microns in greatest diameter averaged 95% of the total and that the length of the crude fiber were less than 5 microns in 95+ percent of the total samples. Crude fibers were used in the cheaper grades of textile and in asbestos shingles, paper, plaster and cement. The milled fibers were less than 5 microns in length in 97% of those counted. The best grades of crude asbestos fiber are used in manufacturing asbestos textiles. Preparation of the asbestos had the highest concentrations which were up to over 100 mppcf averaging 44.26 in preparation and carding; 16.37 mppcf in weaving and mule spinning; & 4.61 in other operations such as gasket making etc. Milled asbestos fiber gave rise to the higher concentrations of crude fiber. Wet methods significantly reduced the counts. Of the 64 workers examined 57 with exposures to asbestos had 14 with asbestosis (25%) and 43 were negative. The most common symptoms were cough and dyspnoea and pleural thickening were found in some (Fulton et al., 1935).

---

around an asbestos mill and also in baboons living near an asbestos mill (Webster, 1963). Animals can serve as sentinel species for use in risk assessment when they are exposed in habitats shared with humans and at similar concentrations. However, no animal species can be expected to respond in exactly the same way as humans and one must take into consideration, when using such data in risk assessment several things including toxic properties of the substance, physiology of the specific animal species and their relationship to potential human exposures (NRC, 1991).

1935 In the study by Lanza et al. (1935), of asbestos textile workers, they found overall 43 percent had fibrosis (lung scaring); 58 percent of workers with 10-15 years exposure; and 87 percent of workers with over 15 years exposure had x-ray changes. Cases of cardiac enlargement were frequently found (later described as Cor-pulmonale); No predisposition to tuberculosis due to asbestos exposure was found; and the authors suggested physical examination at least every 2 years including an X-ray examination of the chest. The authors found the dustiness was greatest in the preparation areas of the five plants studied and that engineering controls reduced the dust by 50% and with further alterations could be reduced by 75% but that it was cost prohibitive to install equipment that would make the environment dust free.

1935 North Carolina makes asbestosis a compensable disease (Shull, 1936).

1936 Asbestosis found in a welder (Jacobson, 1935).

1936 The IHF, first called the Air Hygiene foundation (1936-1941), was founded in 1936, under the auspices of the Mellon Institute. It's membership was comprised of a cross-section of large industrial companies. IHF conducted medical and industrial hygiene surveys, including in the asbestos industry & produced a digest – the Industrial Hygiene Digest which provided health & safety information to members. The IHF meetings were also covered by various news and wire services, such as the Wall Street Journal & The New York Times as well as the Associated Press and United Press International. (McMahon, J.F., 1939 & Castleman, B.I., 1990). The Foundation was labeled by at least by one person, Vandiver Brown, who described to C.J. Stover, the publisher of Asbestos, Dec. 4, 1936 it as "the creature of industry and the one institution upon which employers can rely completely for a sympathetic appreciation of their viewpoint."(Castleman, 1990)

1936 The Journal of the American medical carried an article by Lanza describing asbestosis and its history. (Lanza, 1936).

1936 Studies linking asbestosis with exposure to asbestos (Donnelly, 1936 & Egbert & Geiger, 1936).

1936 McPheeters (1936) described continued exposure to asbestos could increase the fibrosis in existing asbestotics; reported some evidence that asbestosis develops more rapidly in younger persons; no connection to tuberculosis was found; and reduction of the asbestos dust should significantly reduce the incidence of asbestosis.

1936 Shull (1936) discusses his examination of 71 workers dismissed from local asbestos plants in North Carolina beginning in 1934. He concludes that asbestosis is a definite disease entity; one case had only 16 months of exposure; that asbestosis differs for silicosis clinically, pathologically and roentgen-logically; that asbestosis does not predispose to tuberculosis; and he did not observe asbestosis as primarily a progressive disease.

1938 *****United States Public Service Health study of 541 men and women in three asbestos textile factories finding asbestosis dose-response related and later used for setting guidance limits for occupational exposure to asbestos of 5 mppcf (Dreessen et al., 1938).

1939 Asbestosis in a 44 year old man handling asbestos at a chemical plant. His job entailed cutting asbestos into blocks then feeding it into a crusher and packing it into sacks, all while not using a respirator. The authors confirm the observation

by Stewart et al. (1932), that radially arranged bodies, as were in this case, was 'pathognomonic' of the disease (Arnold et al., 1939).

1940 In the Journal of the American Medical Association, Gardner writes on pneumoconiosis. He suggests that the effect of asbestos bodies may well be mechanical and not chemical and reports on the dose-response nature of disease and finds in the dozen or more cases without a history of exposure to asbestos to have asbestos bodies in their lungs. He speculates that if the effects of asbestos bodies are indeed mechanical then there should be no progression because of the rounded nature of the bodies as compared to the rough edges of the asbestos fibers which are freshly inhaled (Gardner, 1940).

1940 Asbestosis in Norway (Wolff, 1940).

1941 Johnstone in his book **Occupational Diseases** discusses asbestosis concluding that irritation by asbestos fibers may be the cause of the disease and not the chemical composition of the fiber. He also concludes that the disease asbestosis is progressive even after the cessation of exposure. He also states that the allowable concentration is 10 mppcf for fibers between sizes of 0.5 to 5 micron (Johnstone, 1941).

1941 Asbestosis in a shipyard Insulator (Kuhn, 1941).

1941 The United States Navy recognizes the need for constant vigilance for the detection of asbestos disease since asbestos is used in the insulating the covers of flanges, valves and high temperature steam turbines. In their initial survey of pipe shop of the New York Naval Yard no asbestos disease was detected, however, the material is wetted with localized exhaust used while respirators are used for the dustiest aspects of the jobs (Brown, 1941). Drinker, a health consultant to the United States Maritime Commission, states that "The pressure to turn out ships is great—it should be, for the need is urgent—and often we must condone practices that we would not accept in peacetime (Drinker, 1943).

1941 Asbestosis in a 38 year old man who was a chemical worker exposed mainly to chrysotile asbestos having used crocidolite asbestos only once during his 10 years of exposure (Schrumpf, 1941).

1942 The first description of typical pleural plaques was by Porro et al., (1942) in a survey of 15 cases from the talc industry.[10]

---

[10] **Notes on Pleural Plaques:** Other reports followed including Siegal et al., (1943) in talc workers exposed to talc dusts containing tremolite asbestos. Siegal et al. also noted after their paper was written that it was reported in the Fifty-Seventh Annual Medical Report of theTrudeau Sanatorium that experimental production of intrapleural adhesions in animals were reported. In the 1950's other reports of pleural calcification and pleural activity were reported in asbestos workers: Smith (1952) tremolite talc; (Jacob & Bohlig, 1955) pleural thickening among a cohort of 343 cases in Dresden Germany; Fehre (1956) observed pleural calcifications thought to be due to inhalation of silica, however, the author concludes they are similar to those observed in persons exposed to asbestos dust; and Frost et al. (1956) observed 22 cases of x-ray changes in 31 laggers surveyed from a trade union in Denmark with 19 having had pleural abnormalities including pleural thickening and calcifications. In a review of 6 studies on the complication of pleural plaques in asbestosis patients, in China, found a range for plaques of from 34.2% to 100% and in an another 6 studies of asbestos workers the prevalence of pleural plaques ranged from 1.3% to 29.8% (Cai et al., 2001).

Calcifications resulting from fibrous dust generally are bilateral and situated on the parietal pleura and probably very small amounts of dust are capable of causing pleural calcifications which appear to be due to mechanical irritation (Kilviluoto, 1960). The plaques are progressive and do cause adverse respiratory symptoms, such as dyspnea (breathlessness) and decrements in pulmonary function while it is more likely that diffuse pleural thickening will cause functional impairment (McMillan and Rossiter, 1982; Sheers, 1979; Rosenstock and Hudson, 1986; Rosenstock et al., 1988). Pleural thickening is considered a marker of past exposures (Hillerdal, 1980). There is evidence that persons with pleural plaques are more likely to develop asbestos-induced parenchymal fibrosis than those without such plaques (Rosenstock, 1994). Further is has been found that, in occupationally exposed persons, that appreciable amounts of fibers were found in their thoracic lymph nodes as well as in pleural plaques (Dodson et al., 1991a & 1991b). Asbestos-induced pleural plaques are the most common finding of the asbestos-related abnormalities (Karjalainen, 2002). Only asbestos and erionite fibers appear to be the only causative agents for the typical pleural plaques with the latency normally several decades. Also, they can result from low exposures which some believe are not an important risk factor for asbestos-induced lung cancer (Karjalainen, 2002). Others believe that there is evidence that individuals with asbestos-induced pleural plaques are at a marked increased risk of developing and dying of lung cancer or malignant mesothelioma.

Fletcher (1972) reported asbestos-exposed shipyard workers diagnosed with pleural plaques were at a 137 percent greater risk from dying of cancer of the lung (16 obs. vs. 6.74 exp.; $p < 0.005$; calculated RR = 2.37, 95% CI: 1.36 - 3.86), none of which had radiological evidence of asbestosis; a 2900 percent increased risk of dying from mesothelioma (3 obs. vs. 0.10 exp.; $p < 0.001$; calculated RR = 30, 95% CI: 6.19 - 87.67) and a 55 percent increase risk of other cancers when compared to the general population of the same age but not occupational exposed to asbestos. The risks were not significant among those without pleural plaques. The workers included a variety of crafts workers. In another study of shipyard workers, Edge (1976) reported that workers with mixed asbestos exposures and pleural plaques (without evidence of pulmonary fibrosis) had a 2.5 times greater risk of developing carcinoma of the bronchus, when compared to the matched controls who had a 1.2 times greater risk without plaques. Also, Edge observed 3 mesotheliomas in those with plaques while none occurred in those with no plaques. Edge (1979) in a later study of shipyard workers found that out of 156 workers with asbestos-induced pleural plaques, but with no other radiographic evidence of pulmonary fibrosis, had 8 deaths from lung cancer compared to 3 in those without pleural plaques, a 2-fold increase and 13 mesotheliomas among those with plaques and 2 in those without plaques, a 6-fold increase and that smoking could not explain the increase in lung cancer in these workers. Edge also observed that if he removed the one mesothelioma occurring within the first 2 years of observation that 7 cases occurred in 2637 man-years of observation for an incidence of 1/377 cases per year.

Hillerdal gives several facts concerning pleural plaques: first, plaques are always more widespread on autopsy than x-ray; Two, in populations without endemic plaques 80-90% of the strictly defined plaques are due to occupational exposures and they can also be found in persons with low-level exposures; third, asbestos bodies are more prevalent in person with pleural plaques; fourth, pleural plaques are related to time after exposure to asbestos than to the dose; fifth, in industrially developed countries 2-4% of all males over the age of 40 usually have plaques; sixth, plaques themselves are usually harmless, but as an indicator of exposure they are indicators of sufficient latency for asbestos-induced cancers, e.g. persons with pleural plaques are twice as likely to develop lung cancer as those without such plaques and those with plaques are more at risk of mesothelioma; seventh, those with pleural plaques, in general, have lower lung function; and finally, persons having high rates of pleural plaques from living in areas of local deposits of asbestos such as tremolite, amosite and crocidolite have a high risk of mesothelioma while those with high rates living in areas of anthophyllite do not (Hillerdal, 2001). In residents of Da-yao, China with environmental exposure to crocidolite pleural plaques were prevalent in 11% of those over 20 years of age and in 20% in those over 40 years old (Luo et al., 2003).

1942 Asbestosis in an aluminum melters who worked wearing an asbestos apron and gloves (Williams, 1942).

1942 Asbestosis was reported among insulators (Holleb & Angrist, 1942).

1944 The industrial journal, *Heating and Ventilation*, published an article discussing dusts, including asbestos, as hazards within the industry including asbestosis and including the statement that no minimal safe concentrations have yet been set up. (Hutchinson, 1944).

1944 The Industial Hygiene Digest (IHD) published two abstracts, one on asbestosis among 132 workers showing 29% with radiological evidence of the disease, some in workers with less than 3 years exposure; second, an abstract by the pioneer cancer researcher, Dr. W.C. Hueper, stating that asbestos was among the chemical and physical agents known to cause occupationally induced cancer (IHD, 1944).

1944 On November 16 the Asbestos Textile Institute was established at a meeting of the University Club in New York City for the purpose: 1) To promote ethical business standards ... 2) To develop standards for the quality of the industry's products etc. 3) To lawfully promote and foster industry policies & 4) To act as a clearing house with respect to the industry manufacturing and other such information (Bettes, 1972).

1946 Asbestosis (3 cases) among pipe coverers, identified in a cross-sectional medical surveillance study, occurring below the United States Public Health Services recommended 5 mppcf (Fleisher et al., 1946).

1946 Asbestosis and the changes in the lung of 126 cases are describe from Finland (Wegelius, 1946).

1946 Asbestosis in 2 cases in Finland from anthophyllite asbestos (Noro, 1946).

1947 Asbestosis in a 58 year old insulator covering boilers and pipes (Kennaway Kennaway, 1947).

1949 Asbestosis in an insulator (Franchini & Canepa, 1949).

1950 Three asbestosis cases reported in Denmark, a country without natural occurring asbestos: one in a shipyard lagger and the other among factory workers making insulation products, brakes and flooring materials (Frost, 1950).

1951 Asbestosis in a 40 year old insulator who had worked as a plumber for 15 years prior to becoming a pipe coverer where he came into contact with asbestos and was then diagnosed with asbestosis after 6 years of work with the Works Progress Administration and then as a shipyard worker (Stoll et al., 1951).

1953 Asbestosis in insulators (van Luyt, 1953; Isselbacher et al., 1953; & Weiss, 1953). Isselbacher et al. (1953) states that the fibrosis is progressive.

1955 McLaughlin raised the issue of preventing end product users and dust diseases when he mentioned sprayed-on asbestos containing materials (McLaughlin, 1955).

Pleural effusions, diffuse pleural thickening and rounded atelectasis are also caused by exposure to asbestos (Tossavinen et al., 1997).

1955 Asbestosis is described as a latent disease with x-ray changes occurring early to the lower lobes of the lung and that improved dust controls will reduce the disease and that asbestosis is a preventable disease (Sander, 1955).

1955 Lynch reports that while conditions are improving the textile mills cases of asbestosis are still occurring (Lynch, 1955).

1955 In a study of 1561 employees of an asbestos company to examine non-occupational respiratory disease 45% of the claims occurred among those working in dusty conditions. This cross sectional study in a sub-set of 708 employees found 7 cases of asbestosis and the author accepts that the fibrosis is due to the mechanical action of the fibers and not their chemical composition (Smith, 1955).

1956 Asbestosis in insulation workers (Ahlborg & Hansson, 1956; Hampe, 1956; Molfino & Sannini, 1956; & Frost et al., 1956).

1956 The annual report of the UK Chief Inspector of Factories mentioned "lagging" (insulation work) as being recognized as hazardous (____, 1956).

1957 Asbestosis in a 48 year old man who was a pipefitter (Marks et al., 1957).

1957 Asbestosis, unlike silicosis, the radiological findings of asbestosis are related to the severity of symptoms with the greater the severity the greater the expected findings on the x-ray. The most common symptoms are again affirmed as that of shortness of breath (dyspnoea) (Thomas, 1957).

1958 Asbestosis in insulators (Pendergrass, 1958 & Van der Schoot, 1958)

1959 Asbestosis in insulators (Hertz & Reinwein, 1959).

1960 Asbestosis in a bricklayer (Whitmore et al., 1960).

1960 Over 20 cases of asbestosis among miners in the Transvaal asbestos mining region of South Africa (Wagner et al., 1960).

1960 Asbestosis in insulators and sprayers (Anderson &Campagna, 1960; Ahlmark et al., 1960; Keal, 1960; Wagner et al., 1960; & Eisenstadt & Wilson, 1960).

1960 Klokov (1960) finds it is necessary to do pulmonary function testing as such changes appear before the appearance of radiological changes.

1961 Asbestosis in insulators (Ambrosi & Cavallo, 1961).

1961 Asbestosis in a shipyard worker (Castleman & Kibbee, 1961).

1961 Asbestosis in carpenter and other asbestos workers (Heard & Williams, 1961).

1961 Asbestosis in a plasterer mixer (Telischi & Rubenstone, 1961).

1961 Asbestosis in a 41 year old male auto mechanic doing automotive undercoating (Brugsch & Bavley, 1961).

1962 Asbestosis in 2 refinery workers one an insulator and the other a foreman (Eisenstadt, 1962).

1962 Asbestosis in hod carrier, iron worker, shipyard worker, bricklayer, carpenter, and insulators (Cordova et al., 1962).

1963 Asbestosis in a 56 year old plumber (Castleman & Kibbee, 1963).

1963 Asbestosis in a 54 year old insulator and in a series of 67 cases of asbestosis 40% occurred in insulators. Dust counts rose well above the existing M.A.C. of 177 particles per cm^3 during mixing and was visible in localized dust clouds and it could be seen that the dust floated in the air for a long time and traveled quite a distance at the one building surveyed. The official M.A.C. was questioned as higher concentrations was not occurring even though asbestosis was still occurring (Farina & Mazzanti, 1963, & Leathart & Sanderson, 1963).

1963 *****As a result of some of these report, the American Medical Association (AMA), Council on Occupational Health, published in the *Archives of Environmental Health*, in August, a whole thesis titled **The Pneumoconioses** in which asbestosis was discussed. This document was to alert physicians throughout the United States of the hazards of dust exposure and disease and how to recognize and treat them. The report was reprinted by the AMA and circulated widely (Mayer et al., 1963).

1963 Asbestosis is discussed in a book on **The Pneumoconioses**, by A. J. Lanza in which it is suggested that "asbestosis bodies" be renamed to "asbestos bodies" since there is no evidence bodies are anything but indicators of exposure and that persons without any signs or symptoms of asbestosis will have them (Smith, 1963).

1963 Asbestosis, of all the pneumoconiosis or dust diseases, would appear to be the most serious because of the very short exposure –months instead of years as is the case with silicosis. Even though measures have been in effect to reduce exposures, asbestosis can still be very high --44.5% in asbestos workers with greater than 20 years exposure and 28% in those with 10-15 years exposure (Public Health, 1963).

1964 *****By 1964 there were more than 700 articles in the worldwide medical literature dealing with health effects associated with asbestos exposure (Ozonoff, 1988).

1964 *****Citing 24 prominent articles on asbestos, the United States Public Health Service (USPHS) embarked on another study of the asbestos industry saying: "There is a great need for further definitive information on the health effect from exposure to asbestos dust and fibers and on safe levels of exposure. Much can be added to the present knowledge in this area by epidemiological information obtainable through the longitudinal study of cohorts, established within the asbestos products manufacturing industry, for which more precise data is kept on the characteristics of the workers as well as on the nature and intensity of pertinent environmental exposures." "A primary weakness in the knowledge now available on the health effects from exposure to asbestos is the lack of epidemiological information on workers at risk over long periods of employment." (Cralley et al, 1964).[11]

[11] **Authors Note:** Because the industries manufacturing or using asbestos had not, themselves, initiated such and analysis, the USPHS embarked on this research to fill in the gaps of knowledge, which had the industries already embarked upon or had accepted their responsibility, would have allowed for prevention plans that would have possibility averted the diseases that the USPHS and other studies would eventually find.

1965 Asbestosis in insulators and brake repair workers[12] (McVittie, 1965).

1965 Asbestosis in insulators (Selikoff, 1965).

1965-1970 Epidemiological studies and case reports along with toxicology studies continued up to and after the passage of the Occupational Safety and Health Act of 1970 which created the National Institute for Occupational Safety and Health (NIOSH) and the Occupational Safety and Health Administration (OSHA) (OSHAct, 1970). The first order of business for promulgating a new recommendation for a standard, by NIOSH and the promulgation of a new standard were for asbestos (see section on Regulations for Asbestos in this Timeline).

II
Cancer of the Lung[13 & 14]

[12] Brake repair workers exposed to asbestos have shown marked pulmonary function loss even in the absence of radiographic asbestosis in both smokers and non-smokers. Even in those young age non-smoking asbestos exposed brake workers both declines in transfer factor and transfer coefficient even without the smoking effect (Erdinc et al., 2003).

[13] **Notes on Lung Cancer:** Asbestosis frequently precluded or was found in conjunction with lung cancer among workers exposed to asbestos (Merewether, 1949; Doll, 1955; Buchanan, 1965). This lead some to speculate that asbestosis was necessary and somehow associated in the etiology of lung cancer among those exposed to asbestos, some attributing this association to the "scar" theory of carcinogenesis. This is not strongly supported for all asbestos-associated lung cancers according to Hillerdal (1994), since he observed that a majority of tumors were squamous cell cancers and not adenocarcinomas. Adenocarcinomas were found most commonly among patients with asbestosis and in the lower lobes of the lung, where asbestosis is most prevalent (Karjalainen, 1994). It is true, however, in some cases of advanced asbestosis, that scar carcinomas may develop as an outgrowth of uncontrolled fibrogenesis, just like they do with usual interstitial pneumonitis (UIP), the typical pathologic lesion in asbestosis (Cullen, 1987). Asbestos exposure appears to increase the risk for all histological types of lung cancer (Karjalainen, 1994). Both those with asbestos exposure and also those with asbestosis have risks of lung cancer higher than found in the general population not exposed to asbestos (Broderick et al., 1992). It is more likely that asbestosis is not a precursor to lung cancer, but that both are independent diseases related with a dose-response from exposure to asbestos, and that cancer of the lung can and does occur in the absence of asbestosis (Roggli et al., 1994; Abraham, 1994; Karjalainen, 1994; Hillerdal, 1994; and Jones et al., 1996). McDonald et al. (1994) have presented epidemiological data showing increased risk of lung cancer in occupations with exposure to asbestos in the absence of radiological evidence of pulmonary fibrosis. Hillerdal (1994), in a well designed study having sufficient statistical power, found lung cancer to occur in patients with bilateral pariental pleural plaques but without radiological evidence of asbestosis. Lung cancer continues to be statistically elevated among asbestos workers under surveillance [SIR 1.14; 95% CI 1.01-1.26] (Koskinen et al. 2003). In a Chinese study of 8 asbestos factory cohorts and 3 mining cohorts that the complication rate of lung cancer among asbestotics ranged from 3.5% to 26.9% (Cai et al., 2001). That exposure levels for carcinogens are safe (including asbestos) is brought into question by the findings that the lungs may accumulate massively more cancer-causing airborne particles than previously thought. The bifurcations within the lung may allow high concentrations of particles to build up as much as 100 times as in the other parts of the lung (Balashazy et al., 2003).

Smoking and Risk of lung cancer increases more than just additive but of a multiplicative nature. Both asbestos and smoking are independently capable of increasing the risk of lung cancer. One of the largest cohorts of asbestos workers to demonstrate this is that of the North American insulators studied by Dr. Selikoff. His co-investigator Dr. E. Cyler Hammond of the American

1935	*****Lynch and Smith (1935) in the United States and Gloyne (1935) in the United Kingdom both associated occupational asbestos exposure with lung cancer. Lynch and Smith reported epidermoid carcinoma of the right lung in a 57 year old man who also had pulmonary asbestosis and who had began as a weaver in an asbestos textile plant in 1913 and died in 1934. Gloyne reported two cases of squamous carcinoma of the lung in women workers, the first age 35 who had spent 8 years as a spinner in a textile plant and survived 9 years after that exposure before death. The second woman was 71 who lived 15 years after two brief periods of 6 months and 13 months working in a mattress factory. Both women also had asbestosis.
1936	Reports appeared in the medical literature of lung cancers occurring in asbestos exposed workers (Egbert and Geiger, 1936 & Gloyne, 1936) .
1938	Reports appeared in the medical literature of lung cancers occurring in asbestos workers Horning reports on the case of a 35 year old woman who worked in an asbestos factory for 9 years and Koelsch reports on 12 cases (Hornig, 1938 & Koelsch, 1938).

Cancer Society (ACS) reported among 12,051 insulation workers with more than 20 years of work experience when compared to a control population from the ACS of 73,763 men both of whose smoking history was known that the RR went up to 53.24 for smoking asbestos insulation workers compared to non-smoking asbestos workers with 5.17 and non-asbestos insulation workers, as controls, of 10.85 (Hammond et al., 1979). In addition, another summary of smoking and asbestos exposure combined, reported the RR for 3 additional studies to be 8.2; 32.7; and 25.7 (Blennerhassett et al., 1995). Asbestosis patients had an Standard Mortality Ratio (SMR) of 15.47 (95% CI: 11.2-20.8) for lung cancer (Morinaga et al., 1993). An analysis of 23 studies on asbestos exposure and smoking shows that asbestos multiplies the risk of lung cancer in non-smokers and smokers by a similar factor and that the combined relationship of exposure to asbestos and smoking can be best described by a multiplicative rather than an additive model (Lee, 2001).

[14] **Notes on the relative risk** for lung cancer has varied from 1.0 (Knox et al., 1968) to 17.6 (Elmes and Simpson, 1971) with an average 9.8 relative risk. The prognosis and treatment of asbestos induced lung cancer is no different than lung cancer having another etiology. It appears that all cell types of lung cancer occur in asbestos workers and that the presence or absence of one cell type cannot be used to prove or disprove and association of asbestos exposure with the lung cancer (Churg, 1985). Since 1997 asbestos has been the leading cause of lung cancer in Japan (Morinaga et al., 2001). Most studies of asbestos workers have been among white males, however, when race is considered black men also are at a higher risk when exposed to asbestos. One study reports an OR of 1.8 (95% CI: 1.03-3.1) for lung cancer in black men, however, when using SEER data from 1988-1992 mesothelioma was higher in white men than black (1.7 vs 0.9/100,000) (Muscat et al., 1998). In a survey of Hungarian workers exposed to asbestos with lung tumors 72 patients (24%) of 297 had cumulative occupational asbestos exposures assessed as below 25 fibre-years (between 0.01 and 23.9 fibre-years) (Mándi et al., 2000). In West Germany, a case-control study reported that the results supported a doubling of the lung cancer risk with 25 fiber-years of exposure and when using a two-phase logistic regression model showed OR increases from 0 to ≤1 fiber-years (0.86; 95% CI: 0.55-1.33; 1 to ≤10 fiber-years (1.33; 95% CI: 0.80-2.33); and 10+ fiber-years (1.94; 95% CI: 1.10-3.43) which are similar to those found by Stayner et al., (1997) and Dement & Brown (1994) (Pohlabeln et al., 2002). A case-referent study of Swedish lung cancer patients found clear evidence for the risk of lung cancer at low-dose levels and that the use of linear extrapolation from high exposure levels may underestimate the risks at low doses. For those exposed at 1-2.49 fiber-years the relative risk (RR) was 2.7 (95% CI: 0.7-9.5) in never smokers and for those smoking >20 cigarettes/day the RR was 80.6 (95% CI: 20.2-322.0) (Gustavsson et al., 2002).

1938 German physicians began calling lung cancer an occupational disease of asbestos workers (Nordman, 1938).

1939 The UK Inspectorate of Factories report in the 1938 Annual Report that 12 cases of lung cancer occurred among 103 cases of fatal asbestosis or 11.6% (HMSO, 1939).

1942 *****Hueper (1942) in his classic book on cancer discusses asbestos related lung cancers.

1943 Fourteen cases of lung cancer or 16% of 92 autopsies on asbestosis case which is much higher than the normal rate in autopsy of 2-6% (Wedler, 1943a)

1943 Three cases of pulmonary cancer in asbestotics: 1st case is a 45 year old male exposed to asbestos for 5 years; 2nd case is a 43 year old male with 20 years exposure & 3rd case in a 49 year old female with an incomplete history of exposure (Homburger, 1943).

1944 *****The American Medical Association talks of asbestos and its relationship to lung cancer (JAMA, 1944).

1947 The Chief Inspector of Factories in Great Britain reported on all of the known cases of asbestosis (235) , in Great Britain, where 13% were due to lung cancer when only 1% of all deaths were due to this cause. Among women, known to have asbestosis, 8% had lung cancer when, at that time very few women smoked and the amount of female lung cancer was quite rare (Merewether, 1949).

1947 Kennaway & Kennaway (1947) reported on lung cancer among asbestos exposed persons.

1948 A 37 year old women who worked in a factory making pipe covers for 7 years was diagnosed 15 years later with pulmonary cancer and asbestosis. The cell type of was of the squamous type and asbestos bodies were found. The author concludes that in contrast with the Kennaway & Kennaway (1947) results of tumors in older men that this case is much younger than the normal pattern for squamous cell carcinoma. The author also states that the question of asbestos exposure and lung cancer is still not completely answered by this one case or the multiple case reports reported previously (Cureton, 1948).

1949 The American Medical Association ran an editorial urging increased attention, by the medical profession, to asbestos exposed workers, because of their risk developing lung cancer (JAMA, 1949).

1949 In 115 deaths from asbestosis 11 males and 6 females had pulmonary cancer or 14.8%, two of the cases were brothers and the mean age at death was 52 with a mean exposure to asbestos of 17.3 years and dying within a mean of 6.6 years after leaving the industry (Wyers, 1949).

1949 Compensation Medicine reports on the relationship of asbestosis and lung cancer (Smith, 1949).

1951 A case of bronchogenic carcinoma in conjunction with asbestosis is reported in a 40 year old man who worked as a pipe coverer (Stroll et al., 1951).

1951 Hueper discusses the relationship between asbestos exposure and asbestos (Hueper, 1951).

1951 In a series of necropsy material 17 in the asbestosis group or 14.1% have had primary neoplasm of the lung or which the rate for males was 19.6% and 9.7% in females (Gloyne, SR, 1951).

1951 Lung cancer, asbestosis & Cor pulmonale reported in a 41 year old man who worked for 12 years in an asbestos mill and was diagnosed two years later (Castleman & Fowne, 1951).

1952 Lung cancer in 8 were identified among 4000 asbestos workers in Canada between 1940 and 1950 (Smith, 1952).

1952 In a report from Switzerland, on occupational diseases caused by asbestos, it is concluded that the "connections and transitions to the formation of lung cancer are concerned, they must definitely be considered positive, because observations in agreement with one another are present" (Sroka, 1952).

1952 Lung cancer in asbestos exposed plumbers, steam fitters and gas fitters (Lew, 1952).

1952 The Encyclopedia Britannica states that asbestos dust is a cause of lung cancer and that "The quantity of this agent [the carcinogen] may be so minute as not to cause apparent irritation during the time of exposure" (Encyclopedia Britannica, 1952).

1953 Two cases of bronchogenic carcinoma and asbestosis, one a 41 year old male asbestos mill worker and a 46 year old contractor's helper cutting and sawing asbestos board to insulate pipes, boilers and refrigerators (Isselbacher, 1953).

1954 Breslow et al. (1954) is one of the first to show that certain occupational groups, in conjunction with cigarette smoking, have higher lung cancer risks. When categorizing persons by occupational groupings they observed steam fitters, boilermakers, and asbestos workers, who worked in these occupational groups exhibited 10 lung cancers compared to 1 in controls (Breslow et al., 1954).

1955 Bonser et al. (1955) indicate that the proof is positive for industrial lung cancer associated with asbestos mining and use. In their own research they found 12 male asbestos textile workers (26.1%) with lung cancer and asbestosis and 2 females (8.7%). They noted that in those with lung cancer the fibrosis tended to be somewhat less than in those with only asbestosis.

1955 In **The Diseases of Occupations**, a classic textbook on occupational medicine a discussion on lung cancer among asbestos workers is given along with the description of a case of lung cancer in a 43 year old woman who had worked as a carder and weaver in an asbestos factory between the ages of fifteen and eighteen and then 25 years later developed the cancer (Hunter, 1955).

1955 *****Epidemiological evidence from a United Kingdom cohort of 113 men who had worked for more than 20 years and were employed before 1930 in an asbestos textile plant was reported by Doll in 1955 which showed a ten-fold excess risk of lung cancer (Doll, 1955). This study has proven to be the pivotal study for the causal association of asbestos exposure and lung cancer.[15]

[15] Criticisms of this paper have been made using data from personal interviews with Dr. Doll and from data supplied to Dr. Castleman. One reviewer of the pre-published paper indicated that data was not produced to support that the lung cancer risk was decreasing among those employed after the 1932 regulations. In fact the rates were going up, according to a UK Government report

1960 The E.I. Du Pont De Nemours & Company sponsored a book published by Lea & Febiger on **Modern Occupational Medicine** which had a chapter by Schepers on Occupational Chest Diseases. In the chapter and section on asbestos Schepers concludes that "[P]ulmonary carcinoma has been observed with such high frequency in employees of the asbestos industry that a causal relationship has been accepted by most authorities." He further discusses the causal association of mesothelioma with asbestos exposure and that even a single month of exposure with no additional exposure can result in asbestosis 30 years later. Finally, Schepers talks about "[C]ases are currently cropping up in men and women who had worked as pipe insulators on liberty ships during the second world war without since having had any further asbestos exposure" (Schepers, 1960).

1965 *****The New York Academy of Sciences held a conference on **The Biological Effect Asbestos** in which lung cancer from exposure to asbestos is discussed with studies showing excess cancers (Selikoff and Churg, 1965).

1966 Hueper (1966) lists 240 cases of lung cancer in asbestosis cases, reported from around the world, 197 in men and 41 in women.

1966 The widely read American/British medical journal *The Lancet* ran an editorial on March 5, 1966 discussing the wide news coverage of the "dangers of asbestos", resulting from the New York Academy of Science Conference and the need for the risks of asbestos to be know and to control exposures (Lancet, 1966).

1966 In a study of asbestos textile workers it was found that 42% with asbestosis had bronchogenic carcinomas and the authors concluded that even thought the modern asbestos factory was better controlled the plant may still play a carcinogenic role even in the absence of asbestosis (O'Donnell et al., 1966).

1966 Carcinoma of the lung has risen from 14.8% to 36.6% by 1966 in a study that compared the finding of Wyers (1949) to the present study (Hill, 1966).

1966 Citing this conference the United States government, program on occupational health of the National Center for Urban and Industrial Health, sponsored a two day seminar, in December of 1966, that outlined the need for expanding the governments role in protecting asbestos exposed individuals. The seminar proceedings stated that "It has been known since the early 1900's that excessive exposure to asbestos gives rise to the disabling pulmonary disease *"asbestosis." More recently, evidence has been developed that the incidence of respiratory tract, and other malignancies in asbestos workers is excessive."* (Brown, 1967).

1967 W. Clark Cooper, former Director of the U. S. Public Health Service's Occupational Health Program and currently with the University of California concludes read a paper before the 19th annual meeting of the American Academy of Occupational Medicine that "That there is an increased incidence of lung cancer in many group of workers exposed to asbestos is a fact". He further states that "Present standards for dust control do not appear adequate to prevent asbestosis over a working lifetime ..." (Cooper, 1967).

1967 The British Medical Journal reports that, even though Doll et al. (1955) report that the attack rates in their study suggest that those workers employed after 1931 do not experience rates above those of the general population, Hammond et al. (1965), in the United States, report death rates among insulation workers to be

---

from 13% between 1924-1946 to 22% between 1947-1954. Actually, the number of lung cancers was twice the number expected in a follow-up of those hired in 1933 or after (Castleman, 2001).

7 times higher than among American white males (Br Med J, 1967).

1968 So much concern was express that some governmental, some industry, some labor, and some academia were jointly embarked on ways to eliminate exposures to asbestos (Brown, 1968; Selikoff, 1968; Jobe, 1968; and Hutchinson, 1968).

1968 J.B. Jobe, Vice President of Johns-Manville Corporation described their need to participate in jointly sponsored studies as ". . . another stride to cope with environmental problems involved with its [industry's] operations and products" (Jobe, 1968).

1968 On October 12, a report appeared in the popular periodical *The New Yorker* magazine on asbestos and its' associated health hazards in a 22 page article tiled *The Magic Mineral.* Now not only the scientific community knew of the hazards of asbestos but now one of the most widely read lay publications was telling its' readership of the dangers faced by persons exposed to asbestos (Brodeur, 1968).

III
Mesothelioma [16 & 17]

[16] **Notes on Mesothelioma:** It is a cancer of the mesothelium, the thin lining of a serous cavity, that covers the major internal organs of the body. The rarity and the fact that this type tumor is strongly associated with exposure to asbestos make it a "signal tumor". This means that it is considered an epidemiological marker for exposure to asbestos (Roglli et al., 1992 & Mullen et al., 1991). Wagner was the first to recognize and report primary pleural tumors in 1870 (Wagner, 1870). Credit is given to Adami for the term mesothelioma in 1909 (Whitaker et al, 1992). The modern concepts concerning the pathology and diagnosis of mesothelioma were set forth in 1931 by Kemperer and Rabin (Kemperer & Rabin, 1931). Gloyne described the migration of fibers to the lymph stream and especially into the mediastinal glands in a person with asbestosis (Gloyne, 1933). It is interesting to note that Hesychius the lexicographer defined asbestosis as stuccoing or plastering and Cooke gave the name asbestosis which now, in addition to asbestosis, "may indeed stucco the pleura or the peritoneum" as well as other organs having mesotheial linings (Hill, 1966). The dose-response relationship for mesothelioma was first shown among textile workers exposed to asbestos and then among gas masks workers, miners and millers and shipyard yard workers (Newhouse & Berry, 1976; Jones et al., 1979; Hobbs et al., 1979; & Sheers & Coles, 1980).

This uncommon tumor, mesothelioma, is now today being reported in almost every major study of persons exposed to asbestos. Some have estimated that pleural mesothelioma occurs with an incidence of 1 for every 2 lung cancers; however, these estimates have generally be related to the overall mortality within specific cohorts of asbestos workers and in some based on cumulative asbestos exposure of 25 or more fiber-years and can be rather misleading either as overestimates or *vis versa* (Mandi et al., 2000). In one analysis the authors have thrown out the three highest and the three lowest ratios and report then a range of ratios for mesothelioma to lung cancer from 1.0 to 5.2, however, they actually through out the 4 lowest so the range is really 0.5 to 5.2 (median 2.4). If they had looked at the entire range it would have a range from 0.3 to 18.5 (median 3.67) (McDonald & McDonald, 1981). Thus the actual ratio does vary amongst studies and any reflection on just the median ratio is misleading. Pleural mesothelioma incidence has been increasing in all asbestos using countries despite control measures put in place since the 1970s (Hemminki & Li, 2003). Peritoneal mesothelioma is a much rarer tumor than pleural, for example in Sweden the male incidence is 10-fold less than for pleural tumors, but in females it is somewhat higher or about ½ that of the pleural tumor. Swedish males have shown no increase in peritoneal mesothelioma since 1985 but in females peritoneal mesothelioma has been steadily increasing and has surpassed the rate of pleural mesothelioma (0.16/100,000) (Hemminki & Li, 2003a). The

National Institute for Occupational Safety and Health in conjunction with The National Center for Health Statistics reports between 1987-1996 that various work groups had extremely elevated PMR's for pleural malignancies such as insulation workers at 23.08 (95% CI 1059-43.80); boilermakes at 15.37 (95% CI 7.68-27.50); plasterers 11.61 (95% CI 3.76-27.13); sheetmetal workers 10.35 (95% CI 6.55-15.54); plumbers, pipefitters and steamfitters 7.02 (95% CI 5.12-9.40) as well as 13 other specific occupations with PMR's of 2 or greater. They also report these occupations taking place in several industries including ship and boat building and repairing with a PMR for pleural tumors of 12.60 (95% CI 8.75-17.52) and petroleum refining with a PMR of 5.76 (95% CI 3.29-9.35). Another 15 industries also had PMR's over 2 with all 95% Confidence Intervals that did not include 1 (NIOSH, 1999). The finding of such a high PMR for ship and boat building and repair is consistent with the study of Tagnon et al. of the shipbuilding in Coastal Virginia which found 61 cases of mesothelioma among white males with a relative risk of 15.7 for the shipyard employees reporting exposure to asbestos compared to 4.9 for shipyard employees who did not report exposure to asbestos (Tagnon et al., 1980).

The ratio of occurrence for mesothelioma in the pleural area to the peritoneal area appears to be associated with the degree of exposure (Newhouse et al., 1972). Among the large occupational exposed groups studied approximately 5-7% of the deaths have been due to mesothelioma (Hammond and Selikoff, 1973; and Selikoff, 1976). In Scotland only 5% of the mesotheliomas gave no history of asbestos exposure, while in Canada this lack of association was higher and the Canadian survey gave the annual incidence of about one per million (Gilson, 1973). Other studies have shown the ranges higher up to 23% (Lieben & Pistawka, 1967). Another estimate has projected that as many as 11% of all asbestos workers' deaths in England will be from mesotheliomas (Newhouse and Berry, 1976). Relative risks (RR) ranged between 2.3 -7.0 with a mean of 4.6 for studies published between 1965 & 1975 (Elmes et al., 1965; Newhouse & Thompson, 1965; McEwen et al., 1970; McDonald et al., 1970; Rubino et al., 1972; Ashcroft, 1973; Hain et al., 1974; Zielhuis et al., 1975; & McDonald & McDonald, 1996). Mesotheliomas association with asbestos exposure has generally been very high, generally over 80% and in those that have not stated such exposures when followed up have shown such exposures (Pinto et al, 1995). Dodson et al. (2000) have shown that 10 to 15% of the mesotheliomas arise in the peritoneal area and that fibers also reach the mesentery and omentum in the peritoneal region (Dodson et al., 2001).

In a 1960 report of abdominal cancers, 8 cases of peritoneal cancers were reported in women, 4 of which were suggested to be primary from the ovary and 4 only of the peritoneum and all of the cases were diagnosed with asbestosis. One case was reported in the same series in a male ventilator cleaner with asbestosis (Keal, 1960). Previously a case of peritoneal cancer had been reported in a 53 year old asbestos worker with asbestosis and asbestos fibers were found in the tumor tissue (Leicher, 1954). Three cases of peritoneal mesothelioma were reported among 36 asbestosis cases and another case of peritoneal mesothelioma was reported in an insulation worker (Konig, 1960 & Van der shoot, 1958). In another series of 72 asbestosis cases four peritoneal cancers were reported, 1 in a male and 3 in females, 2 of which were thought to be primary ovarian cancers (Bonser et al., 1955). Eleven cases of peritoneal mesothelioma were report among 8 men and 3 women between the ages of 38 to 78, with latency periods of 20 to 46 years and exposures between 10 months and 32 years. The authors reported a "remarkable feature" of the cases was the minimal degree of fibrosis in the lungs (Enticknap & Smither, 1964). Peritoneal mesotheliomas continued to be reported among various occupations with exposure to asbestos including: in a 47 year old insulator & a 46 year old insulator (Heard & Rogers, 1961 & Frenkel & Jager, 1961); 3 cases among radiologically confirmed asbestotics (Thomson, 1962); 4 among asbestos textile workers (Mancuso & Coulter, 1963); 17 cases with known asbestos exposures (Hourihane, 1964); a 60 year old former shipyard insulator (Owen, 1964); 3 cases among asbestos textile workers (Mann et al., 1966); and 4 cases among asbestos textile workers (O'Donnell et al., 1966). Newhouse & Thompson (1965) reported 27 peritoneal mesotheliomas in London with both occupational as well as some with domestic exposures.

Mesothelioma continues to remain statistically elevated among asbestos workers as demonstrated in the Finish country-wide screening program of 23,285 men and 930 women between 1990 and 1992 (Standard Incidence Ratio (SIR) 2.77; 95% CI 1.66-4.31) (Koskinen et al., 2003). Mortality data have generally underestimated the mortality from mesothelioma on death certificates as there has not been a specific International Classification of Diseases (ICD) code to allow adequate coding for mortality analysis, but hopefully the new 10 revision of the ICD should address this issue. The new ICD-10 codes for mesothelioma are C45.0 for pleural and C45.1 for peritoneal (ICD-10, 1994). Since it has been generally reported that the incidence of mesothelioma in women is much less associated with asbestos exposure, Steenland et al. (2003) suggest that if take-home asbestos exposure were considered the attributable risks may rise to around 90%.

Other sites of mesothelioma have been reported but not of the same incidence as for the pleural or the peritoneal and their relationship to asbestos exposure needs further analysis. Pericardial mesothelioma has also been reported but it has a very low incidence, as reported in one large autopsy study of less than 0.0022% in and by some estimates is related to about 6% of all mesotheliomas (Kobayashi et al., 2001). Dusting of the pericardium with mixed dusts, including asbestos, was reported in an individual when treated for angina pectoris 15 years earlier (Churg et al., 1978). Also, congenital malignant peritoneal mesothelioma has been observed albeit very rarely, with only three cases documented and their association with asbestos is unclear (Paterson et al., 2002).

[17] **Notes on other malignant diseases:** The most common of which are **gastrointestinal tract cancers** with a relative risk of 0.5 (Meurman et al., 1974) to 3.1 (Mancuso and El-Atar, 1967 and Selikoff, 1974). By the 1960s epidemiological studies suggested exposure to asbestos and the increase in gastrointestinal tract malignancies (Selikoff et al., 1964; Enterline, 1965; and Hammond et al., 1965). The Selikoff et al. (1964) study found stomach, colon and rectum cancer increase three times more than expected (29 vs 9.4; RR = 3.09; CI: 2.07 - 4.43). Among 370 New York-New Jersey asbestos insulation workers 12 stomach, colon and rectal cancers were observed when 3.09 were expected (RR = 3.90; 95% CI; 2.01 - 6.81) (Selikoff, 1974). At the meeting of the New York Academy of Sciences Mancuso (1965) reported, during the discussion of these papers, that he had located 16 additional deaths since his original publication and that five of them were cancers (Mancuso, 1963). They included one of the stomach, one of the colon, and two of the rectum which increased their earlier observation up to 11 gastrointestinal cancers versus 4.55 that would have been expected. Mancuso & El-Attar (1967) reported SMR in the 25-44 year age group of 264 and 1235 after cumulative employment-years of 2.1-7.0 and 7.1-12.0, respectively. Selikoff (1977) found increased rates for cancer of the stomach and esophagus (20 obs. Vs. 6.46 expected [Standard Mortality Ratio (SMR) 3.09; 95% CI: 1.89-4.78]) as he did also for cancer of the colon (23 obs. Vs. 7.64 exp [SMR 3.01, 95% CI: 1.91-4.52].) among the 632 workers, from New Jersey and New York, in his cohort of asbestos insulation workers.[17] In his larger study of 17,800 asbestos insulation workers, from the United States and Canada, Selikoff et al. (1979) reported similar observations for cancer of the esophagus (18 obs. Vs. 7.1 exp.[SMR 2.54, 95% CI: 1.50-4.00]), stomach (18 obs. Vs. 14.2 exp.[SMR 1.27, 95% CI: 0.75-2.00]), and colon and rectum (58 obs. Vs. 38.1 exp.[SMR 1.52, CI: 1.16-1.97]). Others have observed similar results for gastrointestinal cancers among workers exposed to asbestos in various countries (Elmes & Simpson, 1977; Kogan, et al., 1972; & Newhouse, 1973). Schneiderman (1974), then senior statistician for the National Cancer Institute, in his early version of a meta-analysis of the existing literature, up to 1974, concluded that "increased exposure to inhaled asbestos particles leads to increased digestive system cancer". Newhouse & Berry (1979) reported an RR, among male asbestos factory workers with exposure less than 2 years of 2.11 (20 obs. vs. 9.5 exp.; Cal. 95% CI: 1.29-3.250 and greater than 2 years of 2.32 (19 obs. vs. 8.2 exp.; Cal. 95% CI: 1.40-3.62). For females the corresponding SMRs were 2.46 (Obs. 14 vs. 5.7 exp.; Cal. 95% CI: 1.34-4.12) and 3.46 (Obs. 9 vs. 2.6 exp.; Cal. 95% CI 1.58-6.57), respectively. McDonald et al. (1983) reported abdominal cancers in males with 20 years latency and with cumulative dust exposures of 10-<20 mpcf.y of 231.6; 20-<40 mpdf.y 247.0; and 40-<80 mpcf.y of 383.6, respectively. Nine of the 12 deaths reported were from colon and rectum cancers. Enterline et al. (1987) reported on the

mortality of cancer in a cohort of 1074 white male followed to death and found the expected number of deaths from cancers of the stomach, large intestine, and rectum of 30.99 when 43 were observed (SMR 1.43; 95% CI: 1.03 - 1.92) with the SMR for stomach cancer being 180.4 ($p < 0.05$). A dose-response was reported, in a fiber year analysis, for gastrointestinal cancers and years since first exposure increased the SMR rate from less than one during the first 20 years to 231, 273 and 500 after 20-24, 25-29; & 30-34 years from first exposure (Finkelstein, 1984). One of the most recent reviews on the epidemiology of gastric cancer and risk factors (Kelley & Duggan, 2003) points out that methodological problems have cast doubt on the association of asbestos with gastrointestinal cancers, even thought such methodological errors were not discussed and then points to one study to dispute such an association, because after heavy exposure to crocidolite, no excess of gastrointestinal cancers were observed, even though this study itself suffered from a major methodological problem, that being over 25% of the total cohort of 6506 were lost to follow-up (deKlerk et al., 1989).. Albin et al. (1990) reported among asbestos cement workers a RR of 3.4 (95% CI: 1.2-9.5) in those workers with ≥40 f-years/ml for colon and rectum cancer. Among pipe fitters and boilermakers a case-control study reported an OR for colon cancer of 10.7 (95% CI: 1.07-103) (Vineis et al., 1993).

That it was biologically plausible for the fiber to pass through the human gastrointestinal mucosa under conditions of the alimentary canal was shown by Cook & Olson (1979) when they were able to show that sediment in human urine contained amphibole fibers.[17] Asbestos fibers as well as asbestos body formation has been shown in tumor tissue taken in the colons of asbestos exposed workers (Erlich et al., 1991). Reports of gastrointestinal tract cancers associated with asbestos exposure have been reviewed by the World Health Organization (WHO) (1989) in which they have concluded that "overall, there seems that there is a correlation between lung cancer and gastrointestinal cancer rates in occupational cohorts [exposed to asbestos] which is not due to chance" (WHO, 1989). Both the Surgeon General of the United States and the Department of Health, Education and Welfare have concluded that past asbestos exposure can result in an excess of gastrointestinal cancers (Richmond, 1978 & Califoano, 1978).

Frumkin & Berlin (1988) did a meta-analysis of cohort studies to estimate the risk of gastrointestinal cancer mortality. They divided their exposure categories for asbestos exposure into two groups; the first representing heavy asbestos exposure was defined by any cohort having an SMR of 200 or greater for lung cancer and the low exposure category represented by any cohort with an SMR below 200. In the cohort with high exposures to asbestos all of the gastrointestinal cancers, except esophageal cancer, were significantly elevated with 95% confidence limits that excluded 100. For the low exposure cohorts all of the SMRs were close to 100 for gastrointestinal cancers. Homa et al. (1994) report, in their meta-analysis on 20 asbestos exposed cohort, that the summarized SMR for colorectal cancer in those cohorts exposed only to amphibole asbestos to be 1.47 (95% CI 1.09 - 2.00) as compared to those cohorts exposed to chrysotile which was 1.04 (95% CI 0.81 - 1.33). In a recent study, death certificate data was analyzed from 4,943,566 decedents from 28 states in the United States from 1979 through 1990. In the analysis the authors identified 15,524 cases of gastrointestinal cancer among 12 occupational groups having elevated Proportional Mortality Ratio (PMR) for mesothelioma, a sentinel tumor for exposure to asbestos, and found slightly elevated PMR for esophageal (108; 95% CI = 107-110); gastric cancers (110: 95% CI = 106-113); and colorectal cancer (109; 95% CI = 107-110). The authors, from the National Institute for Occupational Safety and Health, conclude that their large death certificate study support an association between asbestos exposure and some gastrointestinal cancers (Kang et al., 1997). Results of a mortality study of textile and cement pipe manufactures between 1933 and 1980 found colon cancers statistically significant (27 obs vs 14.78 exp; SMR 1.83; 95% CI: 1.20-2.66) (Berry et al., 2000).

Stomach cancer was increased among rubber workers who worked in the early production stages of mixing and weighing which the authors concluded may point to the role of either asbestos contaminated talc or carbon black, but their results do not support the causal role of nitrosamines